FACULTY *at the*
SCHMIDT COLLEGE OF MEDICINE

Massimo Caputi, Ph.D. | Professor of Biomedical Science
mcaputi@health.fau.edu

Dr. Caputi's research focuses on the mechanisms that regulate the expression of cellular and viral genes. All living organisms need to tightly control the way genes are ultimately translated into proteins; cancer and several genetic diseases are often the result of the failure of such mechanisms.

Dr. Caputi's research group works to characterize the mechanisms that allow the genes coded by the human genome to be first transcribed into messenger RNAs and then translated into proteins. Since all viruses utilize such cellular machineries to replicate efficiently within human cells, Dr. Caputi and his team have exploited the symbiotic relationship between the HIV virus and the human host to develop novel anti-viral therapies, while simultaneously characterizing the complex mechanisms that control the transcription and splicing of both cellular and viral genes.

More recently, Dr. Caputi expanded his research program to develop novel diagnostic tools utilizing microfluidic chip devices. This technology allows the identification of multiple viruses (HIV, Zika, Dengue, Chikungunya) in point of care settings in a cost effective, quick and accurate manner without the need for complex scientific instruments and highly trained personnel.

Dr. Caputi's research has been continuously funded since 2002 from the National Institute of Allergies and infectious Diseases, a branch of the National Institute of Health and more recently by the Florida Department of Health.

Background

Degree in Biological Science (magna cum laude), University of Trieste, Italy 1992
Ph.D., Molecular Genetics, International School for Advanced Studies, Trieste, Italy1996

Human Frontier Science Program, long term fellow.1997-1999

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Massimo Caputi, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Catherine DeMarino, Michelle L. Pleet, Maria Cowen, Robert A. Barclay, Yao Akpamagbo, James Erickson, Nicaise Ndembe, Manhattan Charurat, Jibreel Jumare, Sunday Bwala, Peter Alabi, Max Hogan, Archana Gupta, Nicole Noren Hooten, Michele K. Evans, Benjamin Lepene, Weidong Zhou, Massimo Caputi, Fabio Romerio, Walter Royal 3rd, Nazira El-Hage, Lance A. Liotta & Fatah Kashanchi. (2018) Antiretroviral Drugs Alter the Content of Extracellular Vesicles from HIV-1-Infected Cells. Scientific Reports. 8, 7653.

Evan Clark, Brenda Nava and Massimo Caputi. (2017) Tat is a multifunctional viral protein that modulates cellular gene expression and functions. Oncotarget. 8(16):27569-27581.

Sean Paz and Massimo Caputi. (2015) SRSF1 inhibition of HIV-1 gene expression. Oncotarget. 6(23):19362-63.

Sean Paz, Michael L. Lu, Hiroshi Takata, Lydie Trautmann and Massimo Caputi. (2015) The SRSF1 RNA Recognition Motifs of are strong inhibitors of HIV-1 replication. J. Virology. 89(12):6275-86.

Sean Paz, Adrian R. Krainer and Massimo Caputi. (2014) HIV-1 transcription is regulated by splicing factor SRSF1. Nucleic Acids Res. 42:13812-13823

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed?term=Caputi%2C%20Massimo%5BAuthor%5D

Current Funding:

- NCI 1R56AI138659-01A1 2019-2020 Highly Sensitive HIV Viral Load Assay For Point-of-care Settings

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Kathleen Guthrie, Ph.D. | Associate Professor of Biomedical Science
kguthrie@health.fau.edu

Neurodegenerative diseases and injuries that damage the adult brain lead to loss of neurons, and ultimately some neural functions. Lost neurons cannot be replaced, though the possibility of using neural stem cells to accomplish this has received wide attention. The Guthrie lab focuses on the brain's olfactory system, which has the unique ability to continuously replace neurons, both peripherally and centrally, under normal conditions, and following injury. Unlike most of the adult brain, the system maintains intrinsic neural stem cells that continuously generate new neurons throughout life. These newborn neurons replace those that are gradually eliminated by normal programmed cell death, resulting in adaptive circuit remodeling as the new neurons integrate and form synaptic connections. By using the adult mammalian olfactory system as a model, we seek to study how these stem cell-derived brain neurons develop, what factors and conditions regulate their survival and functional integration, and how they are impacted by injury, neurodegenerative disease, or genetic mutations that cause neurodevelopmental disorders. Angelman Syndrome (AS) is a devastating genetic disorder on the autism spectrum characterized by intellectual impairment and seizures. The AS mutation eliminates neuronal expression of the ubiquitin ligase Ube3a. The role of this enzyme in normal neuron development, and the pathogenesis caused by its loss, is not well understood. Using genetic mouse models, gene transfer into CNS stem cells, imaging techniques, 3D neuron structural analyses and behavioral assays, our current work tracks the development of new, adult-born neurons in the AS brain to determine what abnormalities arise, when these occur during the maturation/integration process, and how sensory function is impacted.

Background

B.S., Biology, Fort Lewis College, Durango, Colorado 1980

B.S., Chemistry, Fort Lewis College, Durango, Colorado 1982

Ph.D., Psychobiology/Neuroscience and Behavior, University of California, Irvine 1990

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Kathleen Guthrie, Ph.D. | Associate Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Smail S, Bahga D, McDole B, Guthrie, K. (2016) Increased olfactory bulb BDNF expression does not rescue deficits in olfactory neurogenesis in the Huntington's disease R6/2 mouse. Chem. Senses, 41:221-232.

Isgor C, Pare C, McDole B, Coombs P, Guthrie, K. (2015) Expansion of the dentate mossy fiber-CA3 projection in the brain-derived neurotrophic factor enriched hippocampus. Neuroscience, 288:10-23. PMC:4324623. NIHMS652724.

Liu H., Lu M, Guthrie K. (2013) Anterograde trafficking of neurotrophin-3 in the adult olfactory system in vivo. Exper. Neurol. 241:125-137. PMC:3570701

McCollum MH, Leon RT, Rush DB, Guthrie KM, Wei J. (2013) Striatal oligodendrogliogenesis and neuroblast recruitment are increased in the R6/2 mouse model of Huntington's disease. Brain Research, 1518:91-103. PMC3684253

Liu H and Guthrie K. (2011) Neuronal replacement in the injured olfactory bulb. Exper. Neurol. 228:270-282. PMCID: PMC3063445.

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed/?term=Guthrie%20KM%5BAuthor%5D&cauthor=true&cauthor_uid=15882910

Current Funding:

- NIDC 5R21DC016467-02 2018-2020 Role Of UBE3A In Neuronal Maturation And Synaptogenesis In Adult-born Neurons

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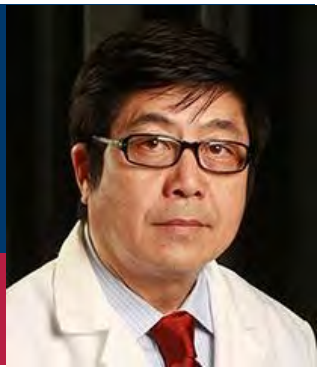

Xupei Huang, M.D., Ph.D. | Professor of Biomedical Science
xhuang@health.fau.edu

The Huang laboratory focuses on revealing mechanisms of myofibril proteins regulating muscle contraction and relaxation in the heart and investigating how a healthy heart becomes a diseased heart due to cardiac protein mutations. Cardiovascular disease is number one killer in the United States. An important question in the field is why a single gene mutation in myofibril protein can cause significant cardiac disorder such as cardiomyopathy even leading to heart failure and early death. For the past 20 years, our laboratory have tried to answer this question by generating and analyzing animal disease models mimicking human diseases and dissecting the dysfunction and disorder at molecular, cellular and whole animal levels. Our laboratory has generated unique mouse models with cardiomyopathy and heart failure caused by myofibril protein mutations.

Using these animal disease models; our laboratory is among the first in the field to demonstrate that myofibril hypersensitivity to calcium is one of the major causes relating to the development of diastolic dysfunction and diastolic heart failure.

Diastolic dysfunction and diastolic heart failure are challenging problems in cardiovascular research since there is no effective medication or treatment for these disorders so far. Searching for medications especially small chemicals to treat diastolic dysfunction is urgent task in the field. Our laboratory, collaborating with other research laboratories, is carrying out high-throughput analysis to identify small molecules in cell-based assays and determine the effectiveness of the leading compounds in animal models. Our results provide insight into the mechanisms underlying the disease development and provide useful clues to find effective treatment for cardiac dysfunction and heart failure.

Background

M.D., Nanjing Medical University, Nanjing, China, 1982

Ph.D., Biochemistry and Biophysics, University of Paris, France, 1992

Secondary post-doctoral fellowship, University of Wisconsin-Madison, 1993-1995 t

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Xupei Huang, M.D., Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Junjun Quan, Zhongli Jia, Tiewei Lv, Lei Zhang, Lingjuan Liu, Bo Pan, Jing Zhu, Ira J. Gelb, Xupei Huang* and Jie Tian* Green tea extract catechin improves cardiac function in pediatric cardiomyopathy patients with diastolic dysfunction. *J Biomed Sci*. 26(1): 32, 2019. DOI: 10.1186/s12929-019-0528-7.

Liu Xiaoyan., Lei Zhang, Daniel Pacciulli, Jianquan Zhao, Changlong Nan, Wen Shen, Junjun Quan, Jie Tian, Xupei Huang. Restrictive cardiomyopathy caused by troponin mutations: application of disease animal models in translational studies. *Front. Physiology*, 7:629, doi: 10.3389/fphys.2017.00629.

Zhang L., C. Nan, Y. Chen, J. Tian, P. Jean-Charles, C. Getfield, X. Wang, X.P. Huang. Calcium desensitizer catechin reverses diastolic dysfunction in mice with restrictive cardiomyopathy. *Archive of Biophys. Biochem*. 573:69-76, 2015.

Yuejin Li, Lei Zhang, Pierre-Yves Jean-Charles, Changlong Nan, Guozhen Chen, Jie Tian, J.-P., Jin, Ira J. Gelb, Xupei Huang. Dose-dependent diastolic dysfunction and early death in a mouse model with cardiac troponin mutations*, J. Mol. Cell. Cardiology*, 62:227-236, 2013.

Li Y., P.Y. Jean-Charles, C. Nan, J. Pinto, Y. Wang, J. Liang, G. Wu, J. Tian, H. Feng, J.D. Potter, J.P. Jin, X.P. Huang. Correcting diastolic dysfunction by Ca^{2+} desensitizing troponin in a transgenic mouse model of restrictive cardiomyopathy. *J. Cell Mol. Cardiol*. 49:402-411, 2010.

Link to Published Work:
https://www.ncbi.nlm.nih.gov/sites/myncbi/1H7Wib4oVgz57/bibliography/51964023/public/?sort=date&direction=descending

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Current Funding:

AHA 19AIREA34380770 2019-2022 (PI) “Correction of diastolic dysfunction and diastolic heart failure in mice with RCM”

FFS-MyoKardia, 19-459A 2019-2022 (PI) “MyoKardia contract”

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Ceylan Isgor, Ph.D. | Associate Professor of Biomedical Science
cisgor@health.fau.edu

Dr. Ceylan Isgor, PhD is trained as a stress neurobiologist and addiction researcher. She has been funded in the past by the NIH/NIDA and Florida Department of Health in studying individual differences in novelty-seeking phenotype (an animal model of human thrill- or sensation-seeking traits), particularly in susceptibility to nicotine craving and relapse. Dr. Isgor expanded her work in the limbic system to delineate intrinsic mechanisms that cause structural rearrangements in synaptic circuits with practical implications for the design of interventions aimed at treating neurological disorders that are characterized by circuit dysfunction. She is recently funded by NIH/NINDS to study epileptogenic changes in forebrain circuits from normal to hyperexcitable states. She uses a transgenic mouse model of brain-derived neurotrophic factor (BDNF) over expression that develops adult-onset spontaneous epilepsy.

Dr. Isgor's lab assess dendritic and spine morphology of neurons that are located in brain circuits that are involved in seizure development and progression mediated by chronic elevations in BDNF in an attempt to understand how slow and progressive rewiring of synaptic circuits can result in full-blown epilepsy. Cortical electroencephalography data obtained from mice show that progression from mild to severe epilepsy is coupled with alterations in sleep architecture that can predict severity of consciousness impairment in upcoming seizure episodes. Dr. Isgor is the Undergraduate Research Liaison for the College of Medicine, and in this role is committed to mutual success of beginning researchers in receiving quality training and exploration of research questions that are pursued in active research programs in the College.

Background

1997, PhD Neuroscience Indiana University, Bloomington IN

1998-2004, Postdoctoral Fellowship, University of Michigan, Ann Arbor MI

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Ceylan Isgor, Ph.D. | Associate Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Bhatti, A., Hall, P., Ma, Z., Tao, R., **Isgor, C**. (2007). Hippocampus modulates the behaviorally sensitizing effects of nicotine in a rat model of novelty-seeking: potential role for mossy fibres. Hippocampus 17(10): 922-933.

Aydin C., Oztan O., **Isgor C**. (2012) Nicotine-induced anxiety-like behavior in a rat model of the novelty-seeking phenotype is associated with long-lasting neuropeptidergic and neuroplastic adaptations in the amygdala: Effects of the cannabinoid receptor 1 antagonist AM251. Neuropharmacology 63(8):1335-45.

Aydin C, Oztan O, **Isgor C**. (2014) Hippocampal Y2 receptor-mediated mossy fiber plasticity is implicated in nicotine abstinence-related social anxiety-like behavior in an outbred rat model of the novelty-seeking phenotype. Pharmacol Biochem Behav 125:48-54.

Isgor C, Pare C, McDole B, Coombs P, Guthrie K. (2015) Expansion of the dentate mossy fiber-CA3 projection in the brain-derived neurotrophic factor enriched mouse hippocampus. Neuroscience 288:10-23

McDole B, **Isgor C**, Pare C, Guthrie K. (2016) BDNF over-expression increases olfactory bulb granule cell dendritic spine density in vivo. Neuroscience 304:146-60

Link to Published Work:
http://www.ncbi.nlm.nih.gov/sites/myncbi/ceylan.isgor.1/bibliography/49714330/public/?sort=date&direction=ascending

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Current Funding:

NIH R21 DC016467 2018-2020 (Co-I) “Role of Ube3a in neuronal maturation and synaptogenesis in adult-born neurons”

NIH R15 NS115049 2019-2022 (PI) “Temporal ontogeny of epileptogenesis in a model of adult-onset, spontaneous seizures”

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Zhongwei Li, Ph.D. | Professor of Biomedical Science and
Director of Faculty Development | *zli@health.fau.edu*

Dr Zhongwei Li's group have been mainly focused on the role of RNases and other enzymes in eliminating oxidatively-damaged RNA molecules which are strongly implicated in the pathogenesis of age-related degeneration. Compared to DNA damage and repair, RNA damage has been much understudied. We now know that RNA oxidation accounts for the majority of nucleic acid oxidation found in all living systems studied. Despite the fact that RNA oxidation causes deleterious effect in protein synthesis and other RNA functions, little is known about the potential cellular mechanisms that reduces and eliminated oxidized RNA. Our publications demonstrate that such mechanisms exist in bacteria as well as in human cells, and they involve RNases and enzymes facilitating RNA degradation.

The second area of research focus in the Li Laboratory is on RNA metabolism in bacteria. We have carried out studies of RNA processing and degradation in Escherichia coli, Mycoplasma genitalium and Yersinia pestis, and has discovered new ribonucleases and novel pathways for RNA processing. Using a genomics approach, Dr. Li and his collaborators have identified numerous genes of Y. pestis that play roles in surviving infection of mammalian cells.

Currently, Dr. Li's research also extends to genomic data analysis to discover genes for RNA metabolism, and to identify gene expression patterns that are related to Alzheimer disease pathogenesis.

Background

B.S. Microbiology, Liaoning University, Shenyang, China 1982

M.S. Microbiology, Microbiology, Chinese Academy of Sciences (CAS), Shenyang, China 1984

Ph.D., Microbiology, Chinese Academy of Sciences (CAS), Shenyang, China 1989

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Zhongwei Li, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Li Z, Malla S., Shin B and Li J (2014) Battle against RNA oxidation: molecular mechanisms for reducing oxidized RNA to protect cells. Wiley Interdiscip. Rev. RNA 5:335-346. PMCID: PMC3991771

Wu J and Li Z. (2008) Human polynucleotide phosphorylase reduces oxidative RNA damage and protects HeLa cell against oxidative stress. Biochem Biophys Res Commun. 372:288-292.

Li Z and Deutscher MP. (1996) Maturation pathways for E. coli tRNA precursors: a random multienzyme process in vivo. Cell 86:503-512.

Li Z, Gong X, Joshi VH and Li M. (2005) Co-evolution of tRNA 3' trailer sequences with 3' processing enzymes in bacteria. RNA 11:567-577.

Oneeb Rehman, Hanqi Zhuang, Ali Muhamed Ali, Ali Ibrahim, Zhongwei Li (2019) Validation of miRNAs as Breast Cancer Biomarkers with a Machine Learning Approach. Cancers (Basel) 2019 Mar; 11(3): 431. Published online 2019 Mar 26. doi: 10.3390/cancers11030431

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed/?term=Li%2C+Zhongwei%5BAuthor%5D%2BFlorida+Atlantic+University

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Marc Kantorow, Ph.D. | Professor of Biomedical Science and
Assistant Dean of Graduate Programs | *mkantoro@health.fau.edu*

The Kantorow laboratory seeks to identify and functionally define novel genetic mechanisms regulating cellular differentiation, homeostasis and disease resistance. A major question in the field is what differentiation and cellular remodeling processes regulate immature stem cell populations to differentiate into functional tissues that could be used for regenerative therapies to treat disease. To answer this important question, our laboratory studies the differentiation and cellular remodeling pathways required to form eye tissues with an emphasis on the eye lens. The transparent function of the lens is dependent on the continuous conversion of undifferentiated lens epithelial cells into mature organelle-free and elongated lens fiber cells. Failure of lens epithelial cells to differentiate into fiber cells results in cataract formation that is a leading cause of world-wide visual disability.

Using molecular genetic approaches to identifying initiators of cellular remodeling in the lens, high-throughput analysis to identify gene expression changes during lens differentiation and epigenetic approaches to identify chromatin modifications required for lens differentiation, we have discovered that oxygen levels regulate lens differentiation through activation of key changes in chromatin confirmations and modifications in association with activation of essential transcription factors. Our results provide insight into those mechanisms required for differentiation and disease states of more complex tissues and we have recently extended our studies towards understanding cellular differentiation in the retina.

Background

B.S. Biology, Cum Laude from Towson State University, Towson, MD 1985

Ph.D., Genetics, George Washington University, Washington, D.C. 1991

Senior Staff Fellow, National Eye Institute NIH 1991-1994

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FAU SCHMIDT COLLEGE OF MEDICINE
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Marc Kantorow, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Lens differentiation is characterized by stage-specific changes in chromatin accessibility correlating with differentiation state-specific gene expression. Disatham J, Chauss D, Gheyas R, Brennan L, Blanco D, Daley L, Menko AS, Kantorow M. Developmental Biology. 2019 Sep 1;453(1):86-104. doi: 10.1016/j.ydbio.2019.04.020. Epub 2019 May 25. PMID: 31136738

BNIP3L/NIX is required for elimination of mitochondria, endoplasmic reticulum and Golgi apparatus during eye lens organelle-free zone formation. Brennan LA, McGreal-Estrada R, Logan CM, Cvekl A, Menko AS, Kantorow M. Experimental Eye Research. 2018 Sep;174:173-184. doi: 10.1016/j.exer.2018.06.003. Epub 2018 Jun 4. PMID: 29879393

Parkin elimination of mitochondria is important for maintenance of lens cell ROS levels and survival upon oxidative stress exposure. Lisa Brennan, Joseph Khoury and Marc Kantorow. Biochimica Biophysica Acta (BBA) Molecular Basis of Disease. 2017 1863)1) 21-32. doi: 10.1016/j.bbadis.2016.09.020.

Identification and ultrastructural characterization of a novel nuclear degradation complex in differentiating lens fiber cells. M. Joseph Costello, Lisa A. Brennan, Kurt O. Gillaland, Snoke Johnson, Marc Kantorow. PloS One. 2017 11(8): e0160785. doi: 10.1371.

Differentiation state-specific mitochondrial dynamic regulatory networks are revealed by global transcriptional analysis of the developing chicken lens. Daniel Chauss, Subhasree Basu, Suren Rajakaruna, Z Ma, Victoria Gau, Sara Anastas, Lisa Brennan, J. Fielding Hejtmancik, A. Sue Menko and Marc Kantorow. Genes, Genomes and Genetics G3 (Bethesda). 2014. 13;4(8):1515-27.

Link to Published Work:
http://www.ncbi.nlm.nih.gov/pubmed/?term=kantarow+m

Current Funding:

- NIH R01 EY029708 2019-2024 (PI) “Hypoxia Regulation of the Eye Lens”
- NIH R01 EY026478 2015-2019 (PI) with Dr. A. Sue Menko (MPI) National Eye Institute, NIH “Repurposing classical death pathways for signalling roles in lens differentiation”

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Andrew V. Oleinikov, Ph.D. | Professor of Biomedical Science
aoleinikov@health.fau.edu

During his tenure in science, Dr. Oleinikov worked in several fields of life sciences including protein biochemistry, molecular and cell biology, autoimmunity, vaccine development, high-throughput approaches, and nanobiotechnology. This wide scientific experience helps him to shape his current research in malaria – one of the most devastating diseases on the planet.
His current research interests include functions of proteins of human parasite Plasmodium falciparum that express on the surface of infected red blood cells, mechanisms of parasite-host interactions, malaria vaccine candidates, molecular mechanisms of low birth weight in placental malaria, and anti-adhesion drugs. In addition, he works on development of tools and technologies for single cell analysis, tissue-on-a-chip, and high throughput approaches, in collaboration with scientists from the College of Engineering, as well as on functional role of a giant endocytic and signaling receptor megalin in placenta.

In addition, he works on development of tools and technologies for single cell analysis, tissue-on-a-chip, and high throughput approaches, in collaboration with scientists from the College of Engineering,as well as on functional role of a giant endocytic and signaling receptor megalin in placenta. Dr. Oleinikov's research resulted in discovery of several novel host receptors and their parasitic ligands. Their interactions lead to adhesion of infected red blood cells to both vascular endothelial cells and cells of immune system – the major events that contribute to severe malaria and often lead to death. Understanding of mechanisms of these interactions and corresponding cellular response will allow for development of new drugs to treat malaria, including immunomodulatory and anti-adhesion drugs. Another aspect of his work (in collaboration with scientists at NIH and other institutions in USA, European and African countries) is host immune response to identify targets of protective immunity, which is an important step in development of vaccines, and ultra-sensitive diagnostics of malaria.

Background

M.S. Engineering Physics/Biophysics, Magna Cum Laude from Peter the Great St. Petersburg Polytechnic University, St. Petersburg, Russia, 1983

Ph.D. Molecular Biology/Biochemistry, Moscow State University, Moscow, Russia, 1989

Post-Docs, University of California, Davis, CA, 1991-1994 and 1994-1996

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Andrew V. Oleinikov, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Ghindilis AL, Chesnokov O, Ngasala B, Smith MW, Smith K, Mårtensson A, Oleinikov AV. Detection of sub-microscopic blood levels of Plasmodium falciparum using Tandem Oligonucleotide Repeat Cascade Amplification (TORCA) assay with an attomolar detection limit. (2019) Scientific Reports, V9: 2901.

Chesnokov O, Merritt J, Tcherniuk SO, Milman N, Oleinikov AV – Plasmodium falciparum infected erythrocytes can bind to host receptors integrins aVb3 and aVb6 through DBLd1_D4 domain of PFL2665c PfEMP1 protein. (2018) Scientific Reports, V. 8:17871.

Lybbert, J. Gullingsrud J, Chesnokov O, Turyakira E, Dhorda M, Guerin PJ, Piola P, Muehlenbachs A, Oleinikov AV. – Abundance of megalin and Dab2 is reduced in syncytiotrophoblast during placental malaria, which may contribute to low birth weight. (2016) Scientific Reports, V. 6, 24508; doi: 10.1038/srep24508

Gullingsrud J, Milman N, Saveria T, Chesnokov O, Williamson K, Srivastava A, Gamain B, Duffy PE, Oleinikov AV – High throughput screening platform identifies small molecules that prevent sequestration of Plasmodium falciparum-infected erythrocytes. (2015) Journal of Infectious Diseases, V. 211 (7), p. 1134–43

Oleinikov AV, Amos E, Frey TI, Rossnagle E, Mutabingwa TK, Fried M, Duffy PE – High throughput functional assays of the variant antigen PfEMP1 reveal a single domain in the 3D7 P. falciparum genome that binds ICAM1 with high affinity and is targeted by naturally acquired neutralizing antibodies. (2009) PLoS Pathogens, V. 5, p. e1000386

Link to Published Work:
https://www.ncbi.nlm.nih.gov/myncbi/andrew.oleinikov.1/bibliography/public/

Current Funding:

- NIH 1R21HD092779 (Du and Oleinikov, MPIs), 2017 – 2020 “Placenta-on-a-Chip Sensing Platform to Study Placental Malaria”
- NIH R21AI137721 (Oleinikov and Nefzi, MPIs), 2018 –2020 “High throughput screening for anti-adhesion drugs against placental and cerebral malaria”

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Ning Quan, Ph.D. | Professor of Biomedical Science
nquan@health.fau.edu

The focus of my lab is how the nervous system and immune system form a combined neuroimmune supra-system. We are interested in understanding how these two systems communicate with each other to modulate each other's function. We use multiple techniques in molecular biology, neuroscience, and immunology to accomplish this goal. This multidisciplinary approach creates an ideal environment for training students on broad biomedical research subjects. Advanced technologies such as FACS analysis, cloning, in-cell Western, patch-clamping electrophysiology, production of transgenic mouse and targeted transgenesis, and behavioral analysis are employed in my laboratory.

Our current research led to the discovery of the euflammatory process which can be used to design vaccine-based induction of immune responses as well as bacterial based cancer therapy. We are also conducting detailed analyses of cell-type specific actions mediated by IL-1R1 using several lines of transgenic animals that we created. This research has led to the identification of specific pathways related to the pathogenesis of various psychopathology caused by CNS inflammation.

Background

1985, BS in Biomedical engineering, Huazhong University of Science and Technology, Wuhan, China.

1991, Ph.D in Neurophysiology, University of Tennessee

1991-1995, Posdoc in Neuroscience, Duke and Emory University

1995-1998, Intramural fellow, National Institute of Mental Health

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Ning Quan, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Xiaoyu Liu, Daniel P. Nemeth, Daniel B. McKim, Ling Zhu, Damon J. DiSabato, Olimpia Berdysz, Gowthami Gorantla, Braedan Oliver, Kristina G. Witcher, Yufen Wang, Christina E. Negray, Rekha S. Vegesna, John F. Sheridan, Jonathan P. Godbout, Matthew J. Robson, Randy D. Blakely, Phillip G. Popovich, Staci D. Bilbo and Ning Quan. Cell-type specific interleukin 1 receptor 1 signaling in the brain regulates distinct neuroimmune activities. Immunity 2019. 50:1-17.

Ling Zhu, Xiaoyu Liu, Daniel P. Nemeth, Damon J. DiSabato, Daniel B. Mckim, Braedan Oliver, Gowthami Gorantla, Olimpia Berdysz, Jiaoni Li, Aishwarya D. Ramani, and Ning Quan. Interleukin-1 causes CNS inflammatory cytokine expression via endothelia-microglia bi-cellular signaling. Brain, Behavior, and Immunity 2019, 81:292-304.

Liu, X., Nemeth, D.P., Tarr, A.J., Belevych, N., Syed, Z.W., Wang, Y., Ismail, A.S., Reed, N.S., Sheridan, J.F., Yajnik, A.R., et al. 2016. Euflammation attenuates peripheral inflammation-induced neuroinflammation and mitigates immune-to-brain signaling. *Brain Behav Immun. 54: 140-148, DIO:* 10.1016/j.bbi.2016.01.018

Qian, J., Zhu, L., Li, Q., Belevych, N., Chen, Q., Zhao, F., Herness, S., and Quan, N. 2012. Interleukin-1R3 mediates interleukin-1-induced potassium current increase through fast activation of Akt kinase. *Proc Natl Acad Sci U S A* 109:12189-12194.

Belevych, N., Buchanan, K., Chen, Q., Bailey, M. and Quan, N. Location-specific activation of the paraventricular nucleus of the hypothalamus by localized inflammation. Brain, Behavior, and Immunity. 24 (2010) 1137–1147

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed/?term=Ning+Quan

Current Funding:

R01 NS116914 (Quan, PI) Neuroinflammation, Neuronal IL-1R1, and Behavior	4/1/2020 - 3/31/2025
R01 MH109165 (Quan, PI) Anxiety, IL-1R1, and Neuroinflammation	12/1/15 - 12/1/20
R33 MH 82118515 (Quan, Co-I) The Role of the Intestinal Microbiome in Anxiety and Depression.	8/28/18 - 8/28/20
R01 NS103785 (Quan, Co-I) Cell-Specific Actions of IL-1 / IL-1R1 Signaling Following Traumatic Brain Injury	12/15/18 - 11/30/23

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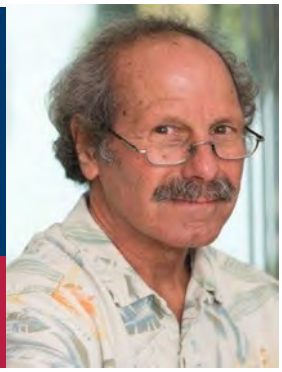

Lawrence Toll, Ph.D. | Professor of Biomedical Science
ltoll@health.fau.edu

For the past 30 years, Dr. Lawrence Toll has studied neurotransmitter receptors and neuropeptides in the brain, primarily opioid and nicotinic systems. Dr. Toll's research on opioid receptors involves collaborations with medicinal and theoretic chemists to identify properties leading to abuse liability, as well as synthesis and characterization of non-addicting analgesics. His main interest is management of pain and drug addiction through pharmacology and new drug discovery. His basic research on opioid and NOP systems, and nicotinic acetylcholine receptors, as well as identification and characterization of endogenous neuropeptides, have opened new avenues of research and identified novel drug targets. In collaboration with medicinal chemists, Dr. Toll seeks to explore basic mechanisms and the biochemical basis of chronic pain and drug addiction, and to identify novel medications for both disorders. Dr. Toll is internationally recognized as the co-discoverer of the neuropeptide "nociceptin", the endogenous ligand for the NOP receptor, the fourth member of the opioid receptor family. This discovery has led to studies of the NOP/nociceptin system and investigation into this system involvement in both pain and reward. Dr. Toll pioneered the idea of a NOP/mu agonist as a potential analgesic with low abuse potential. With respect to nicotinic acetylcholine receptors, Dr. Toll has developed selective ligands as potential smoking cessation medications. Dr. Toll's laboratory has employed a variety of in vitro receptor binding and functional, behavioral, molecular biological, and imaging techniques.

Dr. Toll earned his Ph.D. in Biological Chemistry from UCLA. He stayed at UCLA as a postdoctoral fellow, then accepted a second postdoctoral fellowship in Pharmacology at Johns Hopkins University in Maryland, where he worked for the renowned neuroscientist, Dr. Solomon Snyder. Dr. Toll has over 130 publications and nine patents, issued or pending. His research has been funded by the National Institutes of Health and the Department of Defense.

Background

1978 Ph.D., Biological Chemistry, UCLA, Los Angeles, California

1979-81 Secondary Post-Doctoral Fellowship, Pharmacology, John Hopkins University, Baltimore, Maryland

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Lawrence Toll, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Brunori G, Schoch J, Mercatelli D, Ozawa A, Toll L, Cippitelli A. The influence of neuropathic pain on nAChR plasticity and behavioral responses to nicotine in rats. Pain. 2018 Jun 21. PMID: 29939964

Ozawa A, Brunori G, Cippitelli A, Toll N, Schoch J, Kieffer BL, Toll L. Analysis of the distribution of spinal NOP receptors in a chronic pain model using NOP-eGFP knock-in mice. Br J Pharmacol. 2018 Jul;175(13):2662-2675. PMID: 29582417

Cippitelli A, Brunori G, Schoch J, Armishaw CJ, Wu J, Zaveri NT, Giulianotti MA, Welmaker GS, Toll L. Differential regulation of alcohol taking and seeking by antagonism at α4β2 and α3β4 nAChRs. Psychopharmacology (Berl). 2018 Jun;235(6):1745-1757. PMID: 29572652

Wu J, Cippitelli A, Zhang Y, Debevec G, Schoch J, Ozawa A, Yu Y, Liu H, Chen W, Houghten RA, Welmaker GS, Giulianotti MA, Toll L. Highly Selective and Potent α4β2 nAChR Antagonist Inhibits Nicotine Self-Administration and Reinstatement in Rats. J Med Chem. 2017 Dec 28;60(24):10092-10104. PMID: 29178785

Madariaga-Mazón A, Marmolejo-Valencia AF, Li Y, Toll L, Houghten RA, Martinez-Mayorga K. Mu-Opioid receptor biased ligands: A safer and painless discovery of analgesics? Drug Discov Today. 2017 Nov;22(11):1719-1729. PMID: 28743488

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed/?term=lawrence+toll

Current Funding:

- NIDA R01 2018-2020 DA040882-03 (PI) Npq/Spexin The Endogenous Ligand For The Galanin Receptor 3
- NIDA R41 2018-2022 DA044894-01A1 (PI) The Kappa Partial Agonist Ppl-103 As A Potential Cocaine Abuse Pharmacotherapy
- NIH R01 2018-2022 DA023281-11 (PI) Mixed NOP/mu Compounds and the Involvement of Their Receptors in Analgesia

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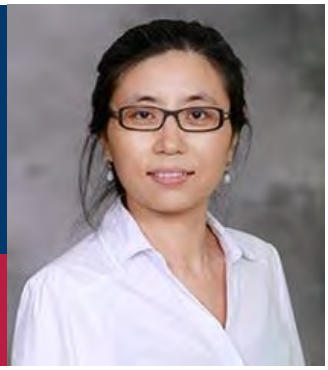

Jianning Wei, Ph.D. | Associate Professor of Biomedical Science
jwei@health.fau.edu

Dr. Jianning Wei's research focuses on understanding the molecular pathogenesis of neurodegenerative diseases using molecular, genetic, biochemical and imaging approaches. Her lab is particularly interested in Huntington's Disease (HD); a devastating fatal neurological disorder caused by a pathological expansion of cytosine-adenine- guanine (CAG) repeats in the huntingtin (htt) gene. HD is often onset at middle age, of which there is a rapid decline, with patients usually dying within 17 years of diagnosis. Dr. Wei is studying molecular signaling pathways affected in this disease by understanding the functions of normal and mutant huntingtin. She also integrates microfluidics into her studies. Using a microfluidic chamber device coupled with electric stimulation, she is able to study activity-dependent axonal transport and molecular changes in primary neurons affected by HD and Alzheimer's disease. Since these diseases have no treatment or cure, it is important that researchers like Dr. Wei continue trying to find therapeutic targets for those suffering.

Dr. Wei is also part of a multi-diciplinary team investigating neuroregeneration utilizing biomedical, mechanical engineering and neurobehavioral expertise to optimize peripheral neuron regeneration in response to different electrical signals. The team is working on a study that explores how feedback loops contribute to the neural regeneration of neural pathways. The goal is to improve the tactile function of amputees fitted with neuroprosthetic limbs, ultimately improving the quality of life of these individuals. Dr. Wei's role is to prepare the peripheral neurons in the microfluidic chamber device and investigate their regeneration properties under different stimulation protocols adapted from amputees.

Dr. Wei's work has been funded by the National Institute of Neurological Disorders and Stroke, National Institute of Biomedical Imaging and Bioengineering and the Florida Department of Health.

Background

B.S., Organic Chemistry, University of Science and Technology of China, Hefei Anhui, P.R.China 1999

Ph.D., Biochemistry, University of Kansas, Lawrence, KS, USA 2003

Fellow, Florida Atlantic University, Boca Raton, FL, USA 2003-2005

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Jianning Wei, Ph.D. | Associate Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Huang, N., Erie, C., Lu, M., Wei, J. (2017) Aberrant subcellular localization of SQSTM1/p62 contributes to increased vulnerability to proteotoxic stress recovery in Huntington's disease. Mol. Cell. Neurosci. 88: 43-52. PMCID: PMC5893379

Erie, C., Sacino, M., Houle, L., Lu, M., Wei, J. (2015). Altered lysosomal positioning affects lysosomal functions in a cellular model of Huntington's disease. Eur J Neurosci. doi: 10.1111/ejn.12957. [Epub ahead of print] PMID: 25997742

McCollum, M., Leon, R., Rush, D., Guthrie, K., Wei, J. (2013) Striatal oligodendrogliogenesis and neuroblast recruitment is increased in the R6/2 mouse model of Huntington's disease. Brain Res. pii: S0006-8993(13)00572-6. doi: 10.1016/j.brainres.2013.04.030. [Epub ahead of print]

Rush, D., Leon, R., McCollum, M., Treu, R., Wei, J. (2012) Palmitoylation and trafficking of GAD65 is impaired in a cellular model of Huntington disease. Biochem J. 442(1) 39-48.

McGreal, R.S., Kantorow, W.L., Chauss, D.C., Wei, J., Brennan, L.A., Kantorow, M. (2012) αB-crystallin/sHSP protects cytochrome c and mitochondrial function against oxidative stress in lens and retinal cells. Biochim Biophys Acta. 820(7), 921-30.

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed/?term=Wei%2C+Jianning%5BAuthor+-+Full%5D

Current Funding:

- NIBIB 5R01EB025819-03 2017-2021 SCH: INT: Virtual Neuroprosthesis: Restoring Autonomy To People Suffering From Neurotrauma
- NINDS 1R21NS111202-01 2019-2021 Dynamic Network Analysis Of Huntingtin Interactome In Response To Cellular Stresses
- 9AZ06 (Florida Department of Health) 2019-2021 Effect of neuronal activity on synaptopathy in Alzheimer's disease using a novel multi-electrode microfluidic platform

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Jang-Yen Wu, Ph.D. | Professor of Biomedical Science
jwu@health.fau.edu

Dr. Wu's research focuses on the fundamental principle underlying normal brain function, as well as brain diseases. Dr. Wu's group focuses on two of the most abundant neurotransmitters in the human brain, namely glutamate and gamma-aminobutyric acid (GABA). In fact, Dr. Wu's group was the first to isolate, purify and characterize the GABA synthesizing enzyme, which laid the foundation for subsequent elucidation of neuronal circuitry using GABA as a neurotransmitter. Because of his groundbreaking work, Dr. Wu was credited as one of the most cited scientists according to the Institute of Scientific Information in 2002.

In addition to basic research, Dr. Wu has also been very active in translational research. Specifically, Dr. Wu used a mechanism-based approach in order to develop therapeutic interventions for various brain diseases including stroke and Parkinson disease. Some of Dr. Wu's discoveries have earned patents by the U.S. Patent Office for the treatment of Parkinson's disease and stroke.

Dr. Wu received his Ph.D. from the University of California Medical School in San Francisco and was later trained under Nobel Laureate, Dr. Paul Boyer, at UCLA. Dr. Wu has served as a faculty member at the City of Hope National Medical Center, Baylor College of Medicine, Pennsylvania State University, Milton Hershey College of Medicine, and at the University of Kansas where he also served as Chairman for the Department of Physiology and Cell Biology before joining FAU as the Senior Schmidt Fellow and Professor in 2002. His research has been supported by a combination of national, state and private funding agencies including the National Institutes of Health, National Science Foundation, Office of Naval Research, State of Florida, American Heart Association, Huntington's Chorea Foundation, the National Multiple Sclerosis Society, and the Marion Merrell Dow Foundation.

Background

B.S., Chemistry, National Taiwan University 1963

Ph.D., Biochemistry, University of California, San Francisco Medical Center 1968

Post-doctoral training, Biochemistry, University of California, Los Angeles 1968-1970

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Jang-Yen Wu, Ph.D. | Professor of Biomedical Science

Representative Publications and Grants

Representative Publications:

Jin, H., Wu, H., Osterhaus, G., Wei, J., Davis, K., Sha, D., Floor, E., Hsu, C.-C., Kopke, R.D. and Wu, J.-Y. Demonstration of functional coupling between GABA synthesis and vesicular GABA transport into synaptic vesicles. Proc. Natl. Acad. Sci. U.S.A. 100: 4293-4298, 2003.

McCollum M., Ma Z., Cohen E., Leon R., Tao R., Wu J.Y., Maharaj D., Wei J. Post-MPTP Treatment with Granulocyte Colony-Stimulating Factor Improves Nigrostriatal Function in the Mouse Model of Parkinson's Disease. Mol Neurobiol. 2010, 41: 410-419. [2 1 Apr 2010, E-Pub ahead of print]

Buddhala, C., Suarez, M., Modi, J., Prentice, H., Ma, Z., Tao., R and Wu, J.-Y. Calpain cleavage of brain glutamic acid decarboxylase 65 is pathological and impairs GABA neurotransmission. PLoS ONE 7(3): e33002. doi:10.1371/journal.pone.0033002 (2012)

Gharibani, P.M., Modi, J., Menzie, J., Genove, R., Ma, Z., Tao, R., Prentice, H., and Wu, J.-Y Mode of action of S-Methyl-N, N-diethylthiocarbamate sulfoxide (DETC-MeSO) as a novel therapy for stroke in a rat model. Mol. Neurobiol., 50 (2): 655-672 (2014).((DOI 10.1007/s12035-014-8658-0, 2014, Feb 28)

Shu, S.-Y., Jiang, G., Zeng, Q.-Y., Wang, B., Li, H.,Ma, L., Steinbusch, H., Song, C., Chan, W.-Y., Chen, X.-H., Wu, Y.-M., Bao, R., Chen, Y.-C. and Wu, J.-Y. The Marginal Division of the Striatum and Hippocampus Has Different Role and Mechanism in Learning and Memory. Mol. Neurobiol. DOI 10.1007/s12035-014-8891- 6 (2014).

Link to Published Work:
https://www.ncbi.nlm.nih.gov/pubmed/?term=Wu%20JY%5BAuthor%5D&cauthor=true&cauthor_uid=31001802